Helicobacter Pylori Infection and Chronic Active Gastritis

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Helicobacter pylori (H. pylori) is an S-Shaped, gram-negative bacillus that recently has been implicated in the pathogenesis of chronic active gastritis and other peptic ulcer disease.[1,2,3,4] These findings have encouraged gastroenterologists to provide a new rationale for patient management, with hope of providing more successful treatment of peptic ulcer disease, particularly gastritis. Therefore, a cooperative diagnostic effort was made at the pathology laboratory of St Francis Medical Center to adopt a simple and reliable method for the identification of H. pylori in tissue sections of endoscopic biopsies of stomach and duodenum.[5] We attempted to estimate the prevalence of H. pylori infection in patients biopsied for upper GI disorders who were refractory to medication. A prevalence of H. pylori infection among different ethnic groups also was studied.

Materials and Methods

Five hundred forty-two consecutive cases were studied by means of gastric and/or duodenal endoscopic biopsies in a 1-year period from October 1989 to September 1990. Each case was examined for histological evidence of gastritis and classified according to the criteria of Whitehead.[6] The presence of increased numbers of lymphocytes and plasma cells in the lamina propria was considered to be indicative of chronic gastritis. Specimens with features of chronic gastritis and a neutrophilic infiltrate in the lamina propria or in glandular epithelium were considered to reflect chronic active gastritis. Atrophic changes and intestinal metaplasia were recorded separately.

Excluded from this study were cases with incomplete clinical information and cases of malignancy or post-operative biopsies taken from the anastomosis sites.

The identification of *H. pylori* was made by studying duplicate slides stained with a modified Wright-Giemsa stain or DiffQuik stain, a product of Baxter Healthcare Corporation in McGraw Park, Illinois. A case was considered to be positive for *H. pylori* only if a small group of S-shaped, bacilli were identified either on or near the mucous surface of the epithelium or in the gastric pits.[5] In equivocal cases, multiple step-sections were searched and double-checked by a second pathologist.

Demographic data of patients were gathered from information provided by the attending clinician from a review of their medical records. Differences between groups were observed by the X2 test: $p<0.05$ was considered significant.

To study the anatomic distribution of the *H. pylori* infection, 5 gastrectomy specimens resected from the patients with duodenal ulcers were sampled according to anatomic areas, such as duodenal cuff, pylorus, antrum and body of the stomach. Gastritis and duodenitis were similarly scored for the presence of *H. pylori.*

Results

***H. pylori* in chronic active gastritis (CAG).**—Our studies showed an overall positivity rate for *H. pylori* of 38.7% (210/542), and an incidence rate for chronic active gastritis of 48.5% (265/542). Of the 210 positive cases, 94% (198/210) revealed chronic active gastritis, representing the highly significant correlation between *H. pylori* colonization and CAG, ($p<0.01$), Table 1.

A small number of cases of chronic non-specific gastritis showed (10/159) *H. pylori* positivity and only 2 of 118 cases of histologically normal gastric mucosa were positive for *H. pylori.*

There were 17 cases of endoscopically diagnosed and biopsy-proven duodenal ulcer cases in our study. Ten of these 17 cases (58%) were positive for *H. pylori.* Organisms were not found in

Table 1.—Correlation Between Inflammation and Presence of *H. Pylori*

Histological category	No. of Patients	*H. Pylori* Positive	Negative	P*
Normal	118	2	116	—
Chronic non-specific gastritis and borderline gastritis	159	10	149	>0.05
Chronic active gastritis	265	198	67	<0.01
Total	542	210	332	

* In comparison with normal tissue

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the necrotic debris which probably originated from the necrotic ulcer crater.

In addition, a total of 79 gastric ulcer cases were included in our study. Of these, 36 cases (45%) were positive for *H. pylori* organisms.

Sex, age and ethnic distribution.—Among all the *H. pylori* positive cases, 108 were in men (51%) and 102 were in women. Our average study population was aged 60, ranging from 21 to 92, Table 2. The highest prevalence of *H. pylori* infection was

Table 2.— Age Distribution of *H. Pylori* Infection

Age group	Male	Female
Less than 30	4	2
31 to 40	11	14
41 to 50	10	12
51 to 60	21	17
61 to 70	31	23
71 to 80	22	22
Older than 81	9	12
Total	108	102

observed in the 61 year to 70 year age group in both men and women. Over 56% of *H. pylori*-positive cases belonged to the over-60 age group. The prevalence of *H. pylori* infection in different ethnic groups is shown in Table 3. In Chinese patients, it was positive in 48.5% (66/136); in Filipinos, 41.8% (62/146); in Japanese, 27.5% (25/91); in Caucasians, 29.6% (27/98); in Hawaiians or part Hawaiians, 45% (16/35); in southeast Asians, 53% (7/13); and in Koreans, 47% (9/19). The differences observed between the 4 larger ethnic groups, Chinese, Japanese, Caucasians and Filipinos, are statistically significant.

Topographic distribution.—Among all of the positive cases, *H. pylori* was most frequently identified in the antrum (79.6% of all positive cases), followed by the body (22%) and cardia (14%). Only 7% of the positive cases were from the duodenum.

There were 142 cases of multiple-site biopsies taken from 2 or more anatomically different locations. Among these cases, 74

Table 3.—Ethnic Distribution of *H. Pylori* Infection

Ethnic group	No. of patients	Hp+	Percentage
Chinese	137	66	48.2
Filipino	147	62	41.1
Caucasian	99	27	27.2
Japanese	92	25	27.1
Hawaiian and Part Hawaiian	35	16	45.7
Korean	19	8	42.1
Southeast Asians	13	6	46.1
Total	542	210	38.7

were all negative and 31 were positive for *H. pylori*. The remaining 37 cases showed mixed results, eg, positive at one site and negative at another. In these, all antral biopsies were positive for *H. pylori*. Of 26 cases with multiple-site biopsies, which included duodenum as one of the sites, all duodenal biopsies were negative for *H. pylori*. Five partial–gastrectomy specimens from patients with perforated or bleeding duodenal ulcers were sampled from different anatomic areas and were scored for the presence of *H. pylori*. The results indicated a predominant antral-pyloric distribution of *H. pylori*.

Discussion

The causative relationship between *H. pylori* and chronic active gastritis was first postulated by Marshall and Warren in 1984.[1] Since then, investigators throughout the world have confirmed that observation and have recognized the pathogenic correlation between *H. pylori* and chronic active gastritis.

Many studies have linked *H. pylori* infection with peptic ulcer disease in general.[2,3,4,7] This has generated new enthusiasm and rationale in patient management and new understanding of inflammatory gastroduodenal diseases.

Hawaii offers a relatively unique opportunity for studying the incidence of diseases in different ethnic groups that is unequaled in any other part of the United States because of the great diversity of races making up its population in a relatively small, confined geographic area.

As clinicians have become increasingly aware of the significance of *H. pylori*, they have gradually increased the number of requests for the identification of *H. pylori*. Therefore, we designed a study to assess the prevalence and the ethnic distribution of this infection in order to get a better understanding of the epidemiology of *H. pylori* infection.

The pathology laboratory at St. Francis Medical Center had adopted a modified Wright-Giemsa stain in 1989 to study *H. pylori* in tissue sections. We found this modified Wright-Giemsa stain to be much simpler and less costly than the Warthin-Starry stain first used by Marshall and Warren.[1] Ours is a reliable and reproducible method that can be used on a routine basis and also allows the best correlation between bacterial identification and tissue diagnosis.

Based on our study, the overall prevalence rate of *H. pylori* infection in our endoscoped patient population was 38.7%, with the prevalence somewhat equal in both men and women. There was a highly significant correlation between *H. pylori* infection and chronic active gastritis. There was also an age-related correlation consistent with the observations of others.[8,9,10,11] Ninety–three percent of *H. pylori*-positive cases were afflicted with chronic active gastritis. Biopsy-proven gastric and duodenal ulcer cases revealed approximately 45% and 58% positivity with *H. pylori*.

Our study did not look into a clear pathognomonic relationship between *H. pylori* infection and peptic ulcer disease. The 5 gastrectomy specimens from patients with perforated or bleeding duodenal ulcers showed a patchy distribution of *H. pylori* in the antral mucosa; the duodenal mucosa near the ulcers were consistently negative for *H. pylori*.

Our results revealed a racial prevalence of *H. pylori* infection that did not correlate well with the known incidence of duodenal ulcer disease reported in Hawaii.[12] Therefore, ulcerogenic etiologic factors appear to be much more complicated and more studies are needed.

Racial differences of *H. pylori* infection have been reported by others[8,9,13] and have raised questions about factors associated with transmission and socioeconomic conditions that cause clustering of the disease and person-to-person, fecal-oral route of transmission.[13,14,15] Preventive measures could be initiated when more epidemiologic information can be obtained.

Summary

A study of 452 consecutive biopsied patients suffering from upper gastrointestinal symptoms demonstrated the following: (1) By histopathologic examination, a total of 210 patients (38.7%) were positive for *H. pylori* and a total of 265 patients (48.5%) had chronic active gastritis. Men and women are equally affected and over 50% of infected patients were older than 60 years of age. Chinese, Filipinos, and Hawaiians had a much higher prevalence as compared to Japanese and Caucasian patients. (2) There is a highly significant correlation between chronic active gastritis and *H. pylori* colonization. Among all *H. pylori* positive cases, 93% had chronic active gastritis. (3) A patchy colonization was noted and more than 75% of positive biopsies were from the gastric antrum. Few positive results were obtained from the biopsies at other sites.

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